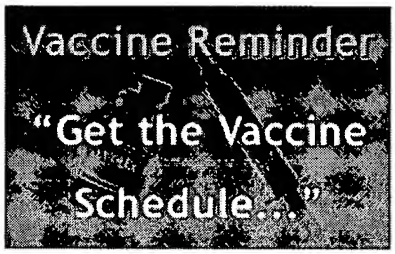
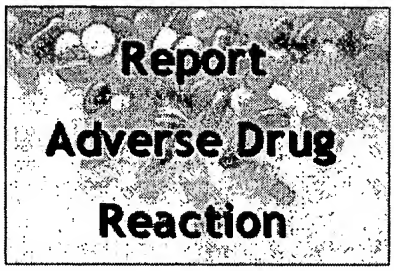
PATTERN OF DECOMPENSATED CIRRHOSIS

In **cirrhosis** , **low** protein, **low albumin**, **reversal of A/ G ratio, low** urea, **low** cholesterol; slight elevation of bilirubin (conjugated), G.G.T.P., S.G.O.T. & alkaline phosphatase are observed. **Low** Ca+ is seen due to hypoproteinemia. **Low** Na+ & Cl are seen due to haemodilution & salt restriction in diet.

BIOCHEMICAL TEST	NORMAL RANGE	PATIENT VALUES		
		LOW	NORMAL	HIGH
TRIGLYCERIDES	35 – 165 mg / dl		50	
BLOOD SUGAR (P.P.) (TRUE GLUCOSE)	Upto 125 mg / dl		80	
F. BLOOD				





Pedi Poll

Now-a-days critical patients are being transferred from nursing homes to higher centres without an RMO accompanying the patient. Is the practice correct?

- No, a doctor should accompany the patient
- Yes, as working manpower is less
- I have not faced this problem.

Vote Results

SUGAR (TRUE GLUCOSE)	65 – 110 MG / DL		75	
L.D.H	40 – 110 IU /			120
S.G.O.T.	7 – 24 IU / L			43
S.G.P.T.	4 – 25 IU / L			40
G.G.T.P	0 – 31 IU / L			90
TOTAL BILIRUBIN	0.2 – 1.0 mg / dl			2.8
CONJ. DIR. BILIRUBIN	0.1 – 0.6 mg / dl			2.0
UNCONJ. BILIRUBIN	0.1 – 0.4 mg / dl			0.8
CHOLESTEROL	125 – 225 mg / dl	115		
TOTAL PROTEIN	6 – 8 Gm / dl	5.8		
ALBUMIN	3.5 – 5.0 Gm / dl	2.2		
A / G RATIO	0.9 – 2.0	0.61		
ALK. PHOSPHATASE	A à 15 – 65 IU / L C à 70 – 150 IU / L			100
CALCIUM	A à 8.5 – 10.5 mg / dl C à 9.5 – 11.0 mg /dl	8.1		
PHOSPHORUS	A à 2.5 – 4.5 mg / dl C à 2.5 – 5.5 mg / dl		4.0	
BLOOD UREA	15 – 45 mg / dl	13		
S. CREATININE	0.5 – 1.5 mg / dl		1.0	
S.URIC ACID	2.1 – 7.4 mg / dl		6.5	
SODIUM	137 – 148m.Eq /	132		

Ped Tools

- Pediatri Calcula
- Drug Ir
- Medical Equipm
- Vaccine Remind
- Adverse Drug Reactio
- Biocher Profile

Calculator

- + Serum Osmolality
- + A-a Grad
- + Bicarbon Base Excess
- + Basal Metabolic R
- + Body Ma Index
- + Body Sur Area
- + Height
- + Weight
- + Head Cir
- + Predict H

	L			
POTASSIUM	3.5 – 5.6m.Eq / L		3.9	
CHLORIDES	99 – 108m.Eq / L	90		
BICARBONATE	23.7 – 31.4m.Eq / L	21		
	A à Adult. C à Child,			

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A combination of serum low albumin and above-average cholesterol level was associated with excess mortality

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Abstract

Background: There is no population-based prospective study concerning the relation between serum albumin and mortality in a non-Western population, and few previous studies included the subgroup analysis stratified by serum cholesterol level.

Methods: A 13.7-year cohort study was conducted on 6,957 males and females aged 30–59 years from 300 randomly selected areas throughout Japan, who participated in the National Survey on Circulatory Disorders in 1980.

Results: In the group with median and above of total cholesterol, one standard deviation (SD) increment of serum albumin (2.6 g/L for males and 2.4 g/L for females) was inversely associated with all-cause mortality for both males and females (relative risk RR = 0.68 and 0.81; 95% confidence interval CI = 0.53–0.87 and 0.68–0.98), and with cancer mortality for females (RR = 0.74; 95% CI = 0.57–0.96); and the lowest category of serum albumin (≤43 g/L) showed the highest cardiovascular mortality for males (RR = 5.04; 95% CI = 1.04–24.5) among the three albumin categories. These relationships were not evident in the group with total cholesterol level below median.

Conclusion: A combination of a low albumin level and above average cholesterol level, even both within the clinical normal range, is associated with excess mortality in the Japanese general population.

Keywords: Serum albumin; Cholesterol; Mortality; Cohort studies

1. Introduction

Several studies have reported higher mortality from all causes [1–8] and higher mortality or morbidity from coronary heart disease [2–4,6,8–13], stroke [14] and cancer [2,4] with decreasing concentrations of serum albumin, a measure of nutritional or acute inflammatory status. To our knowledge, however, there is no prospective study concerning the relation between serum albumin and mortality in a non-Western population. In the meantime, it is well established that a high level of serum total cholesterol (TC) is an important causal risk factor for coronary heart disease and all-cause mortality [15].

Both serum albumin and TC are included in routine serum biochemistry measurement because of convenience and low cost. Furthermore, serum albumin is thought to be an antioxidant because it has binding capacities for free radicals and protects oxidization of serum low-density lipoprotein [16,17]. Few studies have examined the joint and interaction effects of serum TC and serum albumin in identifying persons who are at increased risk of subsequent mortality, and the results of previous studies were controversial [6,10].

Our working hypothesis was that, as with Western populations, serum albumin is a predictor for excess mortality and the relation would be different according to serum TC level, even in a population with low TC levels, such as the Japanese. To investigate this hypothesis, we examined data from a prospective survey of 7,894 persons in the Japanese general population.

2. Materials and methods

2.1. Populations

We used data from the National Integrated Project for Prospective Observation of Non-communicable Diseases

[1] Investigators and members of the research group are listed in references 18, 19, and 20.

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Table 1
Age and age-adjusted means or prevalences of base line characteristics stratified by albumin level at the baseline survey

Risk characteristics	Baseline serum albumin level (stratum mean), g/L						P-value
Male	≤43 (41.8)		44–46 (45.0)		≤47 (48.1)		
Number of participants	806		1,382		874		
Age, years	48.3	(7.5)	43.9	(8.0)	39.6	(7.5)	$P < 0.01$
Cholesterol, mmol/L	4.5	(0.03)	4.9	(0.02)	5.1	(0.03)	$P < 0.01$
Glucose, mmol/L	7.3	(0.07)	7.1	(0.05)	6.9	(0.07)	$P < 0.01$
Systolic BP, mmHg	132.4	(0.66)	134.1	(0.48)	137.1	(0.63)	$P < 0.01$
Diastolic BP, mmHg	80.9	(0.44)	83.0	(0.32)	85.3	(0.42)	$P < 0.01$
BMI, kg/m2	22.4	(0.11)	22.7	(0.08)	23.2	(0.11)	$P < 0.01$
Hypercholesterolemia, %	3.0		6.2		9.5		$P < 0.01$
Diabetes, %	1.5		1.0		0.3		0.05
Hypertension, %	44.4		42.1		39.9		0.18
Daily drinker, %	53.1		48.3		48.1		0.06
Current smoker, %	69.2		64.2		65.0		0.05
Female	≤42 (40.9)		43–45 (44.0)		≥46 (47.0)		
Number of subjects	1,001		1,995		899		
Age, years	45.1	(8.2)	44.0	(8.6)	43.2	(8.9)	$P < 0.01$
Cholesterol, mmol/L	4.6	(0.03)	4.8	(0.02)	5.1	(0.03)	$P < 0.01$
Glucose, mmol/L	7.0	(0.05)	7.0	(0.04)	7.0	(0.06)	0.50
Systolic BP, mmHg	126.7	(0.55)	129.1	(0.39)	133.4	(0.58)	$P < 0.01$
Diastolic BP, mmHg	77.0	(0.35)	78.5	(0.25)	80.9	(0.37)	$P < 0.01$
BMI, kg/m2	23.0	(0.10)	22.8	(0.07)	22.9	(0.11)	0.52
Hypercholesterolemia, %	5.6		6.1		10.9		$P < 0.01$
Diabetes, %	0.9		0.5		0.2		0.10
Hypertension, %	28.2		29.0		35.9		$P < 0.01$
Daily drinker, %	2.0		3.0		1.9		0.10
Current smoker, %	7.3		8.0		9.6		0.18

Abbreviations: BMI, body mass index; BP, blood pressure.
Numbers in parentheses are standard deviation for age and standard error for the other variables.

and Its Trends in the Aged, 1980 (NIPPON DATA80), a national cohort study based on the National Survey on Circulatory Disorders in 1980. Details of the study have been described elsewhere; membership of the research group was as previously published [18–20]. A total of 7,894 community based individuals (3,477 males and 4,417 females) aged 30 to 59 in 300 randomly selected districts were enrolled in the present study, which included a clinical examination and a questionnaire about drinking and smoking. Follow-up was until 1994.

2.2. Mortality data

We used the National Vital Statistics of Japan to determine the cause of death. Permission to use the National Vital Statistics was obtained from the Management and Coordination Agency, Government of Japan. All death certificates issued by physicians were forwarded centrally to the Ministry of Health and Welfare via the public health centers in the area of residency. The underlying causes of death were coded according to the 9th International Classification of Disease. We confirmed those who died in each district by matching data from the National Vital Statistics, using the area, gender, and dates of birth and death as key codes.

2.3. Baseline examination

Nonfasting blood samples were drawn and centrifuged within 60 minutes of collection in 1980. Serum albumin and TC were analyzed in an autoanalyzer (SMA12/60; Technicon, Tarrytown, NY, USA) with the bromcresol green method for albumin and the Lieberman–Burchard direct

Table 2
The number of deaths according to underlying cause of death

Cause of death	ICD9 code	Numbers of death		
		Male	Female	Total
Cardiovascular	393–459	53	32	85
All heart disease	393–398, 410–429	27	18	45
Coronary heart disease	410–414	13	5	18
Stroke	430–438	22	14	36
Other		4	0	4
Cancer	140–208	84	59	143
Stomach	151	20	12	32
Lung	162	21	8	29
Liver	155, 199.1c	15	3	18
Pancreas	157	12	1	13
Breast	174,175	—	9	9
Other		16	24	40
Noncardiovascular, noncancer		54	36	90
Liver disease	570–573	7	3	10
Accident, poisoning, and suicide	800–999	23	14	37
Other		24	19	43
Total		191	127	318

Abbreviations: ICD9, 9th International Classification of Disease.
Among 3,062 males and 3,895 females aged 30–59 years old at baseline during the 13.7-year follow-up. Cause of death: at least five cases in either males or females.

Table 3
The number of deaths and multivariate-adjusted RR (95% CI) for major causes of deat0h according to serum albumin level

Albumin levels (Stratum mean, g/L)	No. of persons	Person-years	All causes: Deaths, no.	RR	95% CI	P-value
Male						
Albumin categories[a]						
≥47 (48.1)	874	11,978	23	1.00		
44–46 (45.0)	1,382	18,829	75	1.41	0.88–2.27	0.16
≤43 (41.8)	806	10,806	93	1.95	1.19–3.19	0.01
P-value, difference between two models[b]						0.14
2.6 g/L (1 SD) increase[c]				0.72	0.61–0.84	<0.01
P-value, difference between two models[d]						0.04
Female						
Albumin categories[a]						
≥46 (47.0)	899	12,394	24	1.00		
43–45 (44.0)	1,995	27,485	64	1.13	0.70–1.81	0.62
≤42 (40.9)	1,001	13,683	39	1.27	0.75–2.14	0.37
P-value, difference between two models[b]						0.07
2.4 g/L (1 SD) increase[c]				0.88	0.74–1.04	0.14
P-value, difference between two models[d]						0.84

[a] The relative risk (RR) was adjusted for age, body mass index, hypertension, diabetes, smoking, drinking, and serum total cholesterol category.
[b] The difference between models with or without categorical interaction term of albumin and cholesterol is compared based on the logarithm likelihood.
[c] The relative risk (RR) was adjusted for age, body mass index, hypertension, diabetes, smoking, drinking, and serum total cholesterol level.
[d] The difference between models with/without linear interaction term of serum albumin and cholesterol is compared based on the logarithm likelihood.
Abbreviations: CI, confidence interval; RR, relative risk.

method for TC at one laboratory (formerly Center for Adults diseases, Osaka; present name, Osaka Medical Center for Health Science and Promotion). Measurement precision and accuracy for serum TC were certified in the Lipid Standardization Program administered by the U.S. Centers for Disease Control and Prevention, Atlanta, GA [21]. Hypercholesterolemia was defined as serum TC being 6.21 mmol/L or greater.

Baseline blood pressures were measured by trained observers using a standard mercury sphygmomanometer on the right arm of seated subjects. Hypertension was defined as systolic blood pressure 140 mmHg or higher, diastolic blood pressure 90 mmHg or higher, use of antihypertensive agents, or any combination of these. Serum glucose was measured by the cupric–neocuproine method [22]. Diabetes was defined as a serum glucose of 11.1 mmol/L or greater, a history of diabetes, or both. Height in stocking feet and weight in light clothing were measured. Body mass index was calculated as weight (kg) divided by the square of height (m). Public health nurses obtained information on smoking and drinking habits, and present and past medical histories.

2.4. Statistical analysis

Because previous studies reported that the relation between serum albumin and mortality varied by gender [4,8,14], gender-specific analyses were performed in the present study. The value of serum albumin was distributed within a narrow range, and cut-points were chosen to give an approximately symmetric distribution across three categories: ≤43 g/L, 44–46 g/L, and ≥47 g/L for males, ≤42 g/L, 43–45 g/L, and ≥46 g/L for females.

Person-years were calculated as the sum of individual follow-up periods until the occurrence of death or to November 15, 1994. Age-adjusted mean values or prevalence of covariates were calculated in each group according to albumin category, and the differences were tested by analysis of covariance or chi-square tests. The association of all-cause and cause-specific mortality to serum albumin was calculated using Cox's proportional hazard model adjusting for age, TC category (below median vs. median and above), hypertension, diabetes, body mass index (BMI), smoking status (never, ex, and current) and drinking status (never, former, occasional, and daily). The highest category was defined as a standard. The model with continuous serum albumin value instead of albumin categories was also examined. The significance of the interaction of serum albumin and TC was tested with an interaction term for either continuous or categorical variables (three albumin categories and the median of serum TC: 4.78 mmol/L for males and 4.76 mmol/L for females) in the multivariate models. A test for difference between two models with or without interaction was performed based on the logarithm likelihood.

Analyses of mortality in relation to serum albumin were also done in the subgroups stratified by the median of serum TC level. Further analyses were repeated excluding deaths within the first 5 years of follow-up. In this analysis, we dealt with early deaths as censored. This analysis could not be performed meaningfully for cause-specific mortality because of the small numbers of each cause-specific death after excluding early deaths. The Statistical Package for the Social Sciences (SPSS Japan, version 10.0J, Tokyo) was used for analysis. All probability values were two-tailed and all confidence intervals were estimated at the 95% level.

Cardiovascular				Cancer				Noncancer, noncardiovascular			
Deaths, no.	RR	95% CI	*P*-value	Deaths, no.	RR	95% CI	*P*-value	Deaths, no.	RR	95% CI	*P*-value
7	1.00			7	1.00			9	1.00		
26	1.55	0.66–3.63	0.31	30	1.50	0.65–3.46	0.34	19	1.20	0.53–2.70	0.66
20	1.32	0.52–3.39	0.56	47	2.04	0.87–4.77	0.10	26	2.40	1.05–5.52	0.04
			0.02				0.27				0.93
	0.84	0.62–1.14	0.27		0.77	0.61–0.98	0.03		0.58	0.44–0.77	<0.01
			0.75				0.66				<0.01
9	1.00			9	1.00			9	1.00		
17	1.30	0.51–3.33	0.58	30	1.34	0.63–2.84	0.44	17	0.79	0.35–1.78	0.57
9	1.35	0.47–3.90	0.58	20	1.57	0.70–3.51	0.27	10	0.86	0.34–2.18	0.75
			0.77				0.08				0.39
	0.81	0.62–1.07	0.14		0.87	0.67–1.14	0.87		1.00	0.72–1.41	0.99
			0.61				0.85				0.74

Of 7,894 participants, a total of 937 were excluded for the following reasons: past history of coronary heart disease or stroke, $n = 123$; missing information at the baseline survey, $n = 131$; and lost to follow-up, $n = 683$, leaving 6,957 participants (3,062 males and 3,895 females) for the analysis.

Approval for the study was obtained from the Institutional Review Board of Shiga University of Medical Science (No. 12-18, 2000).

3. Results

Table 1 shows age-adjusted means or prevalence of the baseline characteristics of all subjects in each albumin group. There were significant differences in the mean values for serum TC and systolic and diastolic blood pressure, with higher values in the higher albumin groups in both genders; the prevalence of hypercholesterolemia was also highest in the highest albumin category. BMI in males, and the prevalence of hypertension in females, also reached statistical significance; they were higher in the higher albumin groups. Mean serum glucose showed an inverse relation across albumin group in males. There was no relation between albumin category and smoking or drinking status.

Total person-years were 95,175 and mean follow-up period was 13.7 years. During the follow-up, there were 318 deaths (191 males and 127 females). The number of cause-specific deaths is shown in Table 2. In this table, numbers of deaths are shown for specific causes where there are at least five deaths: there were 85 deaths from cardiovascular disease (including 18 from coronary heart disease and 36 from stroke), 143 deaths from cancer, and 90 from noncancer, noncardiovascular diseases (including 10 deaths from liver disease and 37 from accident, poisoning, or suicide).

Table 3 shows gender-specific relative risks of cause-specific mortality according to serum albumin level. For males, compared with the highest albumin group, the lowest albumin group showed a significant excess risk of all-cause and noncancer, noncardiovascular mortality (relative risk RR = 1.95 and 95% confidence interval CI = 1.19–3.19; RR = 2.40 and 95% CI = 1.05–5.52, respectively) and, as a continuous variable, the serum albumin level showed a significantly inverse association with all-cause, cancer, and noncancer, noncardiovascular mortality (RR for 1 SD increment of serum albumin = 0.72, 0.77, and 0.58; 95% CI = 0.61–0.84, 0.61–0.98, and 0.44–0.77, respectively) and the differences between two models with or without linear interaction term of serum albumin and TC reached statistical significance ($P = 0.04$ and $P < 0.01$, respectively). There were no significant findings for females. There was no association between serum albumin and subcategories of cardiovascular disease mortality (coronary heart disease, all heart disease, and stroke) in either gender (data not shown in the table).

Table 4 shows gender-specific relative risks of all-cause and cause-specific mortality according to serum albumin level stratified by the median of serum TC. In the group at median and above of TC, for males, the relative risk for all-cause mortality for the lowest albumin group compared with the highest was 3.37 (95% CI = 1.53–7.42) and for the middle group, it was 2.37 (95% CI = 1.13–4.97). In both genders, there was a significant linear inverse association between serum albumin and all-cause mortality; for cardiovascular mortality in males, relative risk for the lowest albumin group compared with the highest was 5.04 (95% CI = 1.04–24.5); serum albumin was not associated with stroke mortality; however, the lowest group of serum albumin compared with the highest shows significant positive

Table 4
The number of deaths and multivariate-adjusted RRs (95% CIs) for major causes of death according to serum albumin stratified by the median of total cholesterol[a]

			All-cause				Cardiovascular			
Albumin levels (Stratum mean, g/L)	No. of persons	Person-year	Deaths, no.	RR	95% CI	*P*-value	Deaths, no.	RR	95% CI	*P*-value
Male										
Below the median of total cholesterol										
Albumin categories[a]										
≥47 (48.1)	350	4,756	14	1.00			5	1.00		
44–46 (45.0)	668	9,048	35	0.80	0.43–1.52	0.50	9	0.55	0.18–1.72	0.31
≤43 (41.8)	498	6,636	62	1.02	0.54–1.94	0.95	10	0.35	0.10–1.21	0.10
2.6 g/L (1 SD) increase[b]				0.77	0.62–0.94	0.01		1.08	0.68–1.74	0.74
P-value, difference between two models[c]						0.09				0.22
Median and above of total cholesterol										
Albumin categories[a]										
≥47 (48.1)	524	7,222	9	1.00			2	1.00		
44–46 (45.0)	714	9,781	40	2.37	1.13–4.97	0.02	17	4.09	0.92–18.1	0.06
≤43 (41.8)	308	4,171	31	3.37	1.53–7.42	<0.01	10	5.04	1.04–24.5	0.04
2.6 g/L (1 SD) increase[b]				0.68	0.53–0.87	<0.01		0.68	0.45–1.03	0.07
P-value, difference between two models[c]						0.05				0.65
Female										
Below the median of total cholesterol										
Albumin categories[a]										
≥46 (47.0)	352	4,822	11	1.00			2	1.00		
43–45 (44.0)	1,005	13,858	27	0.72	0.36–1.46	0.36	5	0.82	0.16–4.31	0.82
≤42 (40.9)	590	8,067	19	0.68	0.32–1.46	0.33	4	0.95	0.16–5.50	0.95
2.4 g/L (1 SD) increase[b]				1.11	0.83–1.47	0.50		0.88	0.47–1.65	0.70
P-value, difference between two models[c]						0.04				<0.01
Median and above of total cholesterol										
Albumin categories[a]										
≥46 (47.0)	547	7,571	13	1.00			4	1.00		
43–45 (44.0)	990	13,627	37	1.54	0.81–2.91	0.41	8	1.63	0.52–5.16	0.40
≤42 (40.9)	411	5,616	20	1.97	0.97–4.02	0.06	5	1.67	0.43–6.55	0.46
2.4 g/L (1 SD) increase[b]				0.81	0.68–0.98	0.03		0.82	0.60–1.11	0.19
P-value, difference between two models[c]						0.17				0.89

Abbreviations: RR, relative risk; 95% CI, 95% confidence interval.

[a] The relative risk (RR) was adjusted for age, body mass index, hypertension, diabetes, smoking, drinking, and serum total cholesterol category.

[b] The relative risk (RR) was adjusted for age, body mass index, hypertension, diabetes, smoking, drinking, and serum total cholesterol level.

[c] The difference between models with/without linear interaction term of serum albumin and cholesterol is compared based on the logarithm likelihood.

Median of total cholesterol was 4.78 mmol/L for males and 4.76 mmol/L for females.

association with all heart disease mortality for males (RR = 8.94; 95% CI = 1.01–78.7; data not shown in the table); the relative risk for coronary heart disease was not calculated because of small numbers. In addition, in females, there was a significantly linear inverse association with cancer mortality (RR = 0.74; 95% CI = 0.57–0.96). In the group with median and above of TC, there was no significant difference between two models with or without linear interaction term of serum albumin and TC for each cause of death.

In the group below median TC, serum albumin level showed significantly inverse association with all-cause and noncancer, noncardiovascular mortality for males (RR = 0.77 and 0.52; 95% CI = 0.62–0.94 and 0.35–0.77, respectively), although these relationships disappeared after excluding deaths due to liver disease (RR = 0.85 and 0.76; 95% CI = 0.69–1.06 and 0.47–1.22, respectively; data not shown in the table). For females, in the analysis of all-cause and cardiovascular mortality, the difference between two models with or without linear interaction term of serum albumin and TC reached statistical significance ($P = 0.04$ and $P < 0.01$, respectively).

Further analysis was performed after excluding deaths within the first five years of follow-up. The results were similar to those shown in Table 4. In the group with median

Cancer				Noncancer, noncardiovascular			
Deaths, no.	RR	95% CI	*P*-value	Deaths, no.	RR	95% CI	*P*-value
5	1.00			4	1.00		
20	1.17	0.43–3.17	0.76	6	0.62	0.17–2.24	0.46
36	1.40	0.51–3.83	0.51	16	1.55	0.45–5.41	0.49
	0.83	0.62–1.09	0.18		0.52	0.35–0.77	<0.01
			0.89				0.14
2	1.00			5	1.00		
10	1.98	0.42–9.35	0.39	13	1.85	0.64–5.35	0.26
11	3.08	0.63–15.1	0.17	10	2.68	0.84–8.54	0.10
	0.67	0.43–1.06	0.09		0.69	0.46–1.04	0.08
			0.67				0.06
5	1.00			4	1.00		
15	0.85	0.31–2.34	0.75	7	0.53	0.15–1.83	0.31
10	0.72	0.24–2.18	0.57	5	0.52	0.14–1.99	0.34
	1.21	0.81–1.81	0.36		1.10	0.65–1.87	1.00
			0.49				0.86
4	1.00			5	1.00		
15	2.05	0.68–6.22	0.21	10	1.00	0.34–2.97	1.00
10	3.34	1.03–10.8	0.04	15	1.14	0.32–4.05	0.84
	0.74	0.57–0.96	0.02		0.98	0.64–1.48	0.91
			0.19				0.25

and above of TC, serum albumin levels in linear analysis for both genders (RR = 0.67, 95% CI = 0.51–0.88 for males; RR = 0.79, 95% CI = 0.64–0.97 for females) and in the lowest compared with the highest albumin group for males (RR = 3.10; 95% CI = 1.34–7.17), showed significant associations with all-cause mortality. These relationships between serum albumin and all-cause mortality were still significant after excluding deaths due to liver disease (RR of the lowest compared with the highest albumin group for males; RR = 2.96, 95% CI = 1.28–6.84; RR per 1 SD increase in linear analyses of serum albumin = 0.68 for males and 0.80 for females, 95% CI = 0.52–0.89 and 0.65–0.99, respectively). In the group with TC level below median, although the serum albumin level showed a significantly inverse linear association with all-cause mortality for males (RR = 0.72; 95% CI = 0.56–0.93), this relation disappeared after excluding deaths due to liver disease. In these analysis after excluding early deaths, we observed no significant difference between two models with or without interaction term of serum albumin and TC both in the group with median and above of TC and that below the median of TC.

4. Discussion

To our knowledge, this is the first prospective population-based study concerning the relation between serum albumin level and mortality in a non-Western population. As seen with Western populations, serum albumin was inversely associated with all-cause, cancer, and noncancer, noncardiovascular mortality for males. Furthermore, in the group with median and above of TC, serum albumin was inversely associated with all-cause mortality for both genders, cancer

mortality for females, and the lowest compared with the highest albumin group had high relative cardiovascular mortality for males, although the confidence interval was wide.

There are only two previous prospective studies on the relation between albumin and mortality or morbidity that include a subgroup analysis stratifying by serum TC level [6,10]. Weijenberg et al. [10] reported an inverse relation between serum albumin and the incidence of coronary heart disease in participants with serum TC levels of 6.5 mmol/L or higher in a 5-year follow-up study of 820 Dutch males aged 64–84 years. This population had a higher level of serum TC (5.88, 6.13 and 6.38 mmol/L in each TC category) than in the present study (median 4.78 mmol/L for males), although the cut-points for albumin were similar (≤43, 44–45, ≥46 g/L for Dutch males; ≤43, 44–46, ≥47 g/L for Japanese males). In contrast, Reuben et al. [6] reported that subjects with low albumin (≤38 g/L) and low TC (≤4.33 mmol/L) had the highest relative risks for 3- and 7-year mortality in a follow-up study of 937 U.S. males and females aged 70 to 79 years. These present results were similar to those of the Dutch study but not the U.S. study. One possible explanation for the different findings in the U.S. study and ours is the differences in age distribution. The present study had a 13.7-year cohort study of middle-aged persons aged 30–59 years old; the U.S. study had a 3- or 7-year cohort study of elderly persons aged 70–79 years. In the U.S. study, low albumin was defined as serum albumin of 38 g/L or less and low TC was defined as serum TC of 4.33 mmol/L or less. Only 2.5% of participants in the U.S. study met both of these criteria, so this combination might indicate a high-risk minority with severe clinical or subclinical disease in the U.S. elderly.

Although any causal mechanism underlying the association of lower serum albumin with higher mortality remains to be elucidated, it is widely accepted that a low albumin level, even within the clinical normal range, may result from subclinical disease [23] and adverse health status [24]. It is also a negative marker of acute-phase inflammatory response [25]. For example, cytokines such as serum interleukin-6, which are increased in acute or chronic inflammatory status, are associated with decreased serum albumin levels [26]. Nelson et al. [12] suggested that albumin level might be a marker of susceptibility to the inflammatory response that resulted from smoking.

Although serum albumin correlated inversely with all-cause and noncancer, noncardiovascular mortality for males in the lower TC group, these relationships disappeared after excluding deaths due to liver disease. Liver disease, such as chronic liver hepatitis or liver cirrhosis, was frequently associated with low serum albumin level, low serum TC level, or both, because of the destruction of the normal hepatocyte in these diseases [27,28]. We did not have information on preexisting liver disease, cancer, and chronic inflammatory diseases, although people with cardiovascular disease were excluded from the analysis; however, the analysis excluding deaths in the first 5 years of the follow-up still showed an inverse association between serum albumin and all-cause mortality, especially in the participants with serum TC at or above the median level.

Serum albumin, the protein itself and possibly also albumin-bound bilirubin, is thought to be an indirect antioxidant because of its specific binding capacities for free radicals [16,17]. It might inhibit vascular injury or progression of atherosclerosis due to oxidized low-density lipoprotein especially at higher TC levels in vivo. The inverse association between albumin and cancer mortality in the higher TC group, which was especially noted in females, may be also due to an indirect antioxidative effect of albumin. We were not able to analyze the relation between serum albumin and site-specific cancer mortality because of small numbers; a larger study in a non-Western population is needed.

A potential drawback of the present study is dilution bias [29], in that the present study was based on only one albumin and TC measurement per person. Furthermore, we did not examine albumin and TC change during the follow-up period. Another limitation is the lack of measurement of oxidized low-density lipoprotein or interleukin-6, measurement of which would have been useful to help understand the relationships among serum albumin, TC, and mortality. Furthermore, we did not make any assessment for renal function or past history of renal disease due to hypertension, diabetes, or chronic nephritis, which may affect serum albumin levels. The prevalence of renal disease should be small, however, because our study population was composed of community dwelling, middle-aged participants who were healthy enough to visit public health centers to undertake an annual health checkup.

In conclusion, a combination of low albumin level and a relatively high TC level, even both within the clinical normal range, was associated with excess mortality in the Japanese general population. Serum albumin is an important marker for long-term mortality, although this may or may not reflect causality. Further cohort studies concerning albumin, TC, and mortality in non-Western populations are indicated.

Acknowledgments

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Hepatitis C Information Central

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Albumin, What is it?

Albumin is a protein manufactured by the liver.

WHAT DOES ALBUMIN DO?

Albumin performs many functions including maintaining the "osmotic pressure" th fluid to remain within the blood stream instead of leaking out into the tissues.

WHAT CAUSES ALBUMIN TO BE TOO LOW?

Liver disease, kidney disease, and malnutrition are the major causes of **low albun** diseased liver produces insufficient **albumin**. Diseased kidneys sometimes lose lar of **albumin** into the urine faster than the liver can produce it (this is termed nephr syndrome). In malnutrition there is not enough protein in the patient's diet for the make new **albumin** from.

WHAT IS THE NORMAL LEVEL OF ALBUMIN?

The normal value depends on the laboratory running the test. Most labs consider r 5 grams per deciliter to be normal.

WHAT HAPPENS IF MY ALBUMIN GETS TOO LOW?

In a healthy person with normal nutrition, the liver will simply manufacture more a will normalize. If **albumin** gets very **low** swelling can occur in the ankles (edema) can begin to accumulte in the abdomen (ascites) and in the lungs (pulmonary edei

HOW DO YOU MAKE YOUR ALBUMIN HIGHER?

The person must return to health. Therefore the underlying disorder must be corre disorder is **cirrhosis** of the liver, the only way to correct **low albumin** is generall liver transplant.

Albumin levels are also dependant on the state of hydration of the body. A persor deficient of water ("dry") because of dehydration will have an artificially **low albur**

Virus Experts Step Closer To Treatment For Hepatitis C, UK
Nine hundred of the world's hepatitis C experts are meeting in Glasgow this week to discuss the latest research into the disease at the 14th International Symposium on Hepatitis C Virus and Related Viruses. Among more than 400 studies being discussed, it

New findings reported from Osaka University, Department of Molecular Virology describe advances in hepatitis C virus immunology
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As reported in the August 21, 2007 edition of the World Journal of Gastroenterology, researchers investigated why some people are able to spontaneously recover from the Hepatitis C virus. This study found the following factors responsible for a person's ability,...

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This returns to normal when the dehydration is corrected. **Albumin** fluctuates so v because it is very sensitive to changes in hydration of the body.

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Albumin

The two serum proteins measured to assess liver function are **albumin** and globulin. Values for total serum proteins range from 6 to 8 g/dl. Of this total, between 52 and 68% (3.5-5g/dl) is **albumin**; the remainder are globulins.

Albumin, produced only in the liver, is the major plasma protein that circulates in the bloodstream. **Albumin** is essential for maintaining the oncotic pressure in the vascular system. A decrease in oncotic pressure due to a **low albumin** level allows fluid to leak out from the interstitial spaces into the peritoneal cavity, producing ascites. **Albumin** is also very important in the transportation of many substances such as drugs, lipids, hormones, and toxins that are bound to **albumin** in the bloodstream. Once the drug or other substance reaches the liver, it is detached from the **albumin** and made less toxic by conversion to a water-soluble form that can be excreted.

A **low** serum **albumin** indicates poor liver function. Decreased serum **albumin** levels are not seen in acute liver failure because it takes several weeks of impaired **albumin** production before the serum **albumin** level drops. The most common reason for a **low albumin** is chronic liver failure caused by **cirrhosis**. The serum **albumin** concentration is usually normal in chronic liver disease until **cirrhosis** and significant liver damage has occurred. In advanced liver disease, the serum **albumin** level may be less than 3.5 g/dl. **Albumin** levels can be **low** in conditions other than liver disease, such as severe malnutrition and some kidney diseases that cause extensive protein wasting. A loss of **albumin** in the urine caused by renal dysfunction (nephrotic syndrome) can cause a decrease in the serum **albumin**. Albuminuria or protein in the urine is a key sign of both renal pathology and pre-eclampsia. Severe burns that damage capillaries and blood vessels cause a huge loss of serum proteins. The increased capillary permeability caused by the burn damage allows a continual leakage of serum proteins out of the vascular system.

When there is inadequate protein intake, the body begins to breakdown muscle to obtain

enough amino acids for the synthesis of serum **albumin**. The United States Department of Agriculture (USDA) recommends that women over age 25 consume 50 gms. of protein daily and men over 25 consume approximately 65 gms. of protein a day. When a patient has liver disease, dietary protein will be decreased to lessen stress on the liver, based on the results of liver function tests. **Albumin** levels do not drop in fasting states or in malnutrition until the condition is severe. A combination of severe illness with prolonged protein deprivation is eventually reflected in a reduced serum **albumin** level. There are no pathological conditions that cause the liver to produce extra **albumin**; thus, an increased rate is a reflection of dehydration.

Instant Feedback:

Albumin is important for maintaining oncotic pressure, and for transporting many substances in the blood.

TRUE or **FALSE**

As the **albumin** level drops in liver disease, there is insufficient oncotic pressure to hold fluids within cells. Fluid moves into the interstitial spaces, causing generalized edema. The collection of fluid in the abdominal cavity, or ascites, may make it extremely difficult for the patient to breathe in a reclining position. A paracentesis or abdominal "tap" may be done to relieve pressure on the diaphragm. A disadvantage of a parencentesis is that proteins are lost when the peritoneal fluid is drained. Diuretics and fluid and sodium restriction may be used to help treat edema.

The exact amounts of **albumin** and each type of globulin are measured through a process called serum protein electrophoresis. This test separates the major proteins in the serum in an electric field to determine the relative concentration of each. Serum protein electrophoresis is a useful test for patients with liver disease because it provides several diagnostic clues. The **Albumin**/Globulin or A/G ratio describes the relationship between **albumin** and globulins. The normal ratio is 1.0 or greater. For example, a patient's **albumin** is 3.5 and the globulin is 2.5, the ratio is 1.4 Although the A/G ratio may still be used, serum protein electrophoresis is now used to compare the amount of **albumin** with globulin.

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Side Effects - Symptoms & Solutions

Hypoalbuminemia (Low Albumin)

What is hypoalbuminemia?

Hypoalbuminemia is a deficit of albumin in the blood, more often seen in elderly patients. Albumin is a protein that is found in the blood.

What causes hypoalbuminemia?

There are many causes of low serum albumin levels. These causes may include:

- Poor nutritional state - you haven't been eating enough protein, or you may be losing protein, usually during a period of illness
- Increased excretion (or loss) of albumin from your body from:
  - Renal (kidney) dysfunction - your kidneys may not work well due to any number of conditions. They may be leaking albumin in the urine, causing **hypoalbuminemia**
  - You may have some form of liver disease, such as **hepatitis**, or cancer in your liver, which may have spread from elsewhere in your body that causes you to lose albumin, thus resulting in **hypoalbuminemia**.
  - Certain heart conditions - such as congestive heart failure, or pericarditis - may cause you to have low albumin levels in your blood
  - Problems with your stomach - including inflammatory bowel disease, or lymphoma, can cause **hypoalbuminemia**
  - Other forms of cancer or conditions- such as sarcoma or amyloidosis - can cause **hypoalbuminemia**
  - Side effects from medications can cause **hypoalbuminemia**
  - Infections - such as tuberculosis - can cause **hypoalbuminemia**

What are some symptoms and side effects of hypoalbuminemia to look for?

- You may not have any symptoms, unless your blood albumin levels are significantly lowered. In this case, you may not be eating very well. You may have swelling that is all over your body, or swelling in one part of your body (such as your legs)
- You may have muscle weakness, fatigue, or cramps
- You may have a poor appetite, and may not be eating well. Even people who take in a lot of protein in their diet may still have low albumin levels in their blood.
- If you have liver problems that may have caused your **hypoalbuminemia**, you may notice that your abdomen is swollen with fluid (called, ascites).

Things you can do to treat hypoalbuminemia:

- Follow your healthcare provider's instructions regarding raising your blood albumin level. Treatments of low albumin levels are based on correcting the underlying cause.
- Make sure you tell your doctor, as well as all healthcare providers, about any other medications you are taking (including over-the-counter, vitamins, or herbal remedies). These can cause interactions with other medications.
- Take all of your medications as directed
- Speak with your doctor or healthcare provider about which diet is right for you. Depending on the cause of your **hypoalbuminemia**, he or she may suggest a different type of diet. For example, if you have low albumin levels in your blood due to improper nutrition, you may be encouraged to eat high- protein foods. If your **hypoalbuminemia** is due to liver dysfunction, you may be placed on fluid restriction, and a special diet. Discuss this with your healthcare provider.
- Avoid alcohol, as alcohol can cause your symptoms of **hypoalbuminemia** to worsen (especially with liver disease)
- Follow all of your healthcare provider's recommendations for follow up blood work and laboratory tests to monitor your **hypoalbuminemia**.
- If you experience symptoms or side effects of your therapy, especially if severe, be sure to discuss them with your health care team. They can prescribe medications and/or offer other suggestions that are effective in managing such problems.

Drugs or treatments that may be prescribed by your doctor for hypoalbuminemia:

Treatment of low albumin levels (**hypoalbuminemia**) is based on correcting the underlying cause. The medications that your doctor or healthcare provider may prescribe vary greatly depending on the cause of your **hypoalbuminemia**.

Return to list of Blood Test Abnormalities

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Hypoalbuminemia

From Wikipedia, the free encyclopedia

Hypoalbuminemia is a medical condition where levels of albumin in blood serum are abnormally low. It is a specific form of hypoproteinemia.

Hypoalbuminemia *Classification & external resources*	
ICD-9	273.8 (http://www.icd9data.com/getICD9Code.ashx?icd9=273.8)

Albumin is a major protein in the human body, making up about 60% of total human plasma protein by mass. Many hormones, drugs, and other molecules are mostly bound to albumin in the bloodstream and must be released before becoming biologically active.

Albumin is synthesized in the liver, and low serum albumin may be indicative of liver failure or diseases such as **cirrhosis** or chronic hepatitis. **Hypoalbuminemia** can also present as part of the nephrotic syndrome, in which protein is lost in the urine due to kidney damage. Low albumin levels can be an indicator of chronic malnutrition.

Hypoalbuminemia may cause generalized edema (swelling) via a decrease in osmotic pressure.

The serum albumin level is part of a standard panel of liver function tests. Levels below 3.5 grams per deciliter are generally considered low.

External links

- *med/1116 (http://www.emedicine.com/med/topic1116.htm)* at eMedicine
- DDB *31324 (http://www.diseasesdatabase.com/ddb31324.htm)*
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- Treating **Hypoalbuminemia** In Critically Ill Patients (http://www.medscape.com/viewarticle/484802)

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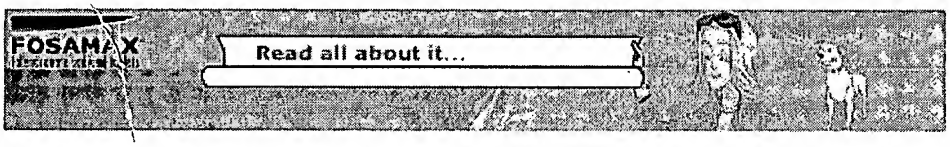
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Evaluation of Common Liver Problems

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Abstract

Diseases of the liver and abnormalities of liver enzyme levels are among the most common reasons for gastroenterology consultation for surgical patients. Although the differential diagnosis of liver disease is exceedingly broad, the majority of patients will have one of several common disorders. A familiarity with the major disorders affecting the liver and the approach to patients with liver disease is therefore useful for clinicians from all disciplines. This article reviews common liver diseases, including the viral hepatitides; the interpretation of liver enzyme abnormalities; and the relationship between common medications and liver disease. Although an exhaustive knowledge of hepatology is impractical for most clinicians, a practical approach to common liver diseases is a necessity for all. (J Am Podiatr Med Assoc 94(2): 149-156, 2004)